LESIONS OF THE PRIMITIVE FIBRILS OF THE VOLUNTARY MUSCLES IN PROGRESSIVE MUSCULAR ATROPHY AND IN LEAD PARALYSIS. The following is the report of a communication made to the Soc. de Biologie, Feb. 19, by MM. Renaut and Debove, as given in the *Gaz. des Hopitaux*, No. 22.

It has long been clinically known that voluntary muscles affected with progressive atrophy preserve their sensibility and electro-contractility to the last. Their voluntary contractility likewise continues, though unable, it is true, to producc movement through the weakness of the muscle. It persists, nevertheless, in its integrity in so far as we consider it as a physiological act, independent of the useful effect produced by its contraction.

In the paralysis from chronic lead poisoning, on the other hand, the muscular atrophy is quickly accompanied by loss of contractility and of electric sensibility. Consequently the muscle ceases to contract under the influence of the will, (Vulpian and Reymond). We propose to demonstrate in this communication the anatomical differences between these two kinds of muscular atrophy, and to explain by them the diversity of the symptoms observed.

When we isolate and color by puro-carminate of ammonia or purpurine the primitive bundles of the voluntary muscles affected with simple progressive muscular atrophy, we observe (1) that the muscular substance has only diminished its volume so that its bundles, compared with healthy muscles, become slender and sometimes filiform. The sarcolemma is preserved in its integrity, and lies closely on the surface of the bundle. Under it we see numerous nuclei, many of which are arranged in twos and threes, and one lodged in the crossed fibres of the muscular substance.

This last, though diminished in volume, is nowhere interrupted. In specimens properly prepared, the longitudinal and transverse striae are perfectly preserved. On the fibres closely observed, however attenuated they may be, we recognize with the greatest clearness the thick disk, the clear space which separates it from its adjoining one, and the smooth disk which traverses it.

From this it follows that the inflammatory processes of which the primi tive fibre is the seat, lead generally in the atrophied muscles to the segmentation and multiplication of nuclei on the one hand, and on the other to diminution of the contractile substance in volume, leaving its structure intact. This last substance may be reduced in the atrophied primitive bundle to a few juxtaposed fibres, unable, therefore, to perform any appreciable work, but still ready to contract singly under the influence of differ ent muscular excitants, natural or otherwise.

The histological changes in muscles met with in progressive muscular atrophy afford us, therefore, an exact account of the phenomena observed in the living patient, that is, the preservation of the voluntary contractility, the electric sensibility and contractility, which subsist up to the exact moment when the muscular substance has disappeared in the primitive bundles.

We have both successfully noted the multiplication of muscular nuclei

in progressive muscular atrophy and the lesions of the muscles in lead paralysis. We cannot here enter into the history of this question. We propose to do this completely in a future work. But if we compare the primitive bundles of voluntary muscles affected with progressive atrophy, and by the atrophic paralysis due to the slow action of lead on the organism, it is easy to recognize that in this last the lesions are altogether different from those that characterize progressive muscular atrophy. The extensor muscles under the influence of lead, offer all the characters of a sub-acute myositis, such as is produced by introducing and leaving a seton in the flesh. The primitive bundles are no longer cylindrical but moniliform. Here and there the nuclei are proliferated and accumulate under the sarcolemma, swelling it out in a lump. At this point the muscular substance is strangulated or completely cut off in such a way that the nuclei distend the tube of sarcolemma, and above and below them there is seen a fragment of contractile substance. This vegetation of the muscular nuclei occurs here and there, in such a way that the muscular substance is divided into segments and the continuity of the bundles is interrupted.

Hence we can understand how a muscle so injured is incapable of contraction, either under the influence of the will, or from the application of different physiological excitants, since it is made up of disconnected fragments having no action on the tendinous extremities.

We will not here review the different methods in which this fragmentation is effected, this disassociation and resorption of the muscular substance in primitive bundles in lead paralysis. These details formed the subject, in fact, of a memoir presented to the Soc. de Biologie the past year by one of us. We have intended only to show in this communication that the physiological differences observed in the living person, in the muscles affected with progressive atrophy or lead paralysis, have their *raison d'être* in relatively considerable anatomical lesions, and result from two different modes of degeneration.

The Symptomatic Importance of Deviation of the Head. At the meeting of the Soc. de Biologie, Feb. 8, (rep. in *Gaz. de Hopitaux*,) M. Lepine related the case of a woman, suffering from Bright's disease, brought to the hospital in a state of coma, hemiplegic with contracture of the left side and with the face also turned to that side. Usually in hemiplegias the face is turned toward the side of the cerebral lesion, the unparalyzed side of the body. In this case the reverse was the case, which led M. Lepine to think that the lesion would be found in the protuberance or pons.

The autopsy confirmed this supposition; there was found a very small hemorrhagic clot of the size of a bean, a little to the right of the middle line of the pons. Beside this was found a minute point, appearing at first sight like a miliary aneurism, but which turned out, on microscopic examination, to be an old sanguine extravasation. In spite of the most minute examination M. Lepine could discover no trace of miliary aneurisms. The capillaries were atheromatous.